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Department of Gastroenterology, Department of Clinical Immunology,
Medical University of Lublin

HALINA CICHOŻ-LACH, MARIA SŁOMKA,
KRZYSZTOF CELIŃSKI, ANNA BĄCZEK

Level of serum cytokines in biliary gastritis and erosive gastritis with Helicobacter pylori coinfection

Cytokines are substances secreted by cells or group of cells in order to cause specific changes of behaviour of the other cells. Cytokines operate locally. Some of them are present in serum. Recent studies have established that cytokine mediators, when released from circulating or tissue-fixed macrophages, not only function as endocrine mediators, but also act in a paracrine fashion to regulate metabolic and immune function near their tissue of origin (4, 6). There are many papers presenting serum cytokines or their receptors levels in different gastrointestinal diseases, but few of them value cytokines level in different types of gastitis. Thus it seems interesting to examine this problem.

The aim of this study was evaluation of serum cytokines level in patients with biliary and erosive gastritis with or without *Helicobacter pylori.*

MATERIAL AND METHODS

50 patients were admitted to this study: 20 with biliary gastritis (13 women and 7 men), 20 with erosive gastritis (11 women, 9 men) and 10 men as the control group. Mean age was 43.2 years (between 21 and 74). Diagnosis of biliary gastritis and erosive gastritis was based on gastroscopy examination. In all patients serum levels of interleukin-8 (Il-8), interleukin-6 receptor (Il-6R), interleukin-2 receptor (Il-2R) were evaluated by Predicta ELISA (enzyme-linked immunosorbent assay) procedure of Gezyme Diagnostics. In each group half patients were infected by *Helicobacter pylori*, and the second half were uninfected. *Helicobacter pylori* infection was evaluated with the urease test.

RESULTS

Mean level of Il-8 in patients with biliary gastritis and *Helicobacter pylori* infection was 80 ±14 pg/ml. It was higher than in those in the control group (42±8 pg/ml), and more increased than in uninfected patients (62±12 pg/ml). Differences were statistically significant ($p<0.05$). In erosive

gastritis accompanied by *Helicobacter pylori* infection mean level of Il-8 was 48±9 pg/ml and was higher than in uninfected patients for which the level was normal, such as in the control group (Tab. 1).

Tab.1. Mean serum level (±SD) of Il-8 in patients with and without infection *Helicobacter pylori*

	Helicobacter pylori (+)	*Helicobacter pylori* (-)
Gastritis biliaris	80±14 *	62 ±12 *
Gastritis erosiva	48±9	40±6
Control group	42±8	36±11

* Statistically significant differences.

Tab. 2. Mean serum level (±SD) of Il-6R in patients with and without *Helicobacter pylori* infection

	Helicobacter pylori (+)	*Helicobacter pylori* (-)
Gastritis biliaris	120±36 *	102±32 *
Gastritis erosiva	88±21 *	64±19
Control group	62±19	60±13

* Statistically significant differences.

Tab. 3. Mean serum level (±SD) of Il-2R in patients with and without *Helicobacter pylori* infection

	Helicobacter pylori (+)	*Helicobacter pylori* (-)
Gastritis biliaris	80±13	77±19
Gastritis erosiva	78±17	80±21
Control group	77±16	79±14

Serum Il-6R levels in biliary gastritis with infection by *Helicobacter pylori* were higher than in biliary gastritis without infection (mean level was 120pg±36 pg/ml and 102±32 pg/ml). These values were statistically significant vs. the control group ($p<0.05$). In erosive gastritis accompanied by

Helicobacter pylori mean level of Il-6R (88±21 pg/ml) was higher than in the control group (62±19pg/ml), $p<0,05$. In patients without *Helicobacter pylori* mean Il-6R was 64±19 pg/ml, and was similar to the control group (Tab. 2). The mean Il-2R level in patients with biliary gastritis, to compared erosive gastritis, and to control group with or without *Helicobacter pylori* infection, was the same. No significant differences were found (Tab. 3).

DISCUSSION

Interleukines Il-1, Il-2, Il-6, Il-8 are pro-inflammatory cytokines. *Helicobacter pylori* infection stimulates Il-8 secretion in gastric epithelial cells. Il-8 is a strong mediator of inflammatory process (7). Receptors of cytokines make link in information transfer routes between cells.

H. pylori infection stimulates defence mechanisms of immunological system. Specific antibodies are secreted locally in mucosa. Cells are activated, which is accompanied by the presence of a variety of inflammation mediators, especially cytokines (5).

H. pylori is the main responsible factor for inflammatory changes in the gastric mucosa. In the majority of cases, chronic active gastritis which is accompanied by infiltration with multinuclear cells, lymphocytes, and plasmatic cells in superficial and glandular coat is detected.

The other gastritis factors are chemical agents like bile, nonsteroid anti-inflammatory drugs (NSAID). They cause erosive or biliary gastritis (2). It is very difficult to determine relations between erosive or biliary gastritis, serum level of interleukines and *H. pylori* infection. It was found that Il-8, Il-6R levels in biliary gastritis increased when patient was *H. pylori* infected. These results are in agreement with those published (7). Serum level of Il-8 is higher in patients with *H. pylori* than in noninfected (7, 1). In biliary gastritis the levels of the cytokines of interest were higher than in erosive gastritis. It is supposed that gastric mucosa damaging factors, among them duodenogastric reflux which cause chemical gastritis, may play some role here (3).

No influence of *H. pylori* infection was noticed on Il-2R levels in patients with biliary gastritis and erosive gastritis. These levels were normal in all groups of patients. Further investigations are needed to explain these results. In our study we found the levels of Il-8, Il-6R higher in biliary gastritis than in erosive gastritis. Particularly significantly high levels of Il-8, Il-6R were observed in gastritis accompanied by *H. pylori* infection.

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SUMMARY

The aim of this study was evaluation of serum cytokines level in patients with biliary and erosive gastritis. 50 patients were admitted to this study: 20 with biliary, 20 with erosive gastritis and 10 as the control group. In all patients serum levels of Il-8, Il-6R, Il-2R were evaluated. In each group half patients were infected by *Helicobacter pylori,* and the second half were uninfected. Mean level of Il-8 in patients with biliary gastritis was higher than in those in the control group, and was more increased in *Helicobacter pylori* infected patients than in uninfected ones. In erosive gastritis accompanied by *Helicobacter pylori* infection mean level of Il-8 was higher than in uninfected patients for which the level was normal. Serum Il-6R levels in biliary gastritis with infection by *Helicobacter pylori* were higher than in biliary gastritis without infection. These values were statistically significant vs. the control group. In erosive gastritis accompanied by *Helicobacter pylori* mean level of Il-6R was higher than in the control group, but was normal in patients without *Helicobacter pylori.* We noticed no influence of *Helicobacter pylori* infection on Il-2R levels in patients with biliary gastritis and erosive gastritis. These levels were normal in all groups of patients. In conclusion, in biliary gastritis we found the levels of Il-8, Il-6R higher than in erosive gastritis. Particularly significantly high levels of Il-8, Il-6R were observed in gastritis accompanied by *Helicobacter pylori* infection.

Poziom surowiczych cytokin w żółciowym i nadżerkowym zapaleniu żołądka ze współistniejącą infekcją *Helicobacter pylori*

Celem pracy była ocena poziomu surowiczych cytokin u pacjentów z żółciowym i nadżerkowym zapaleniem żołądka oraz ze współistniejącą infekcją *Helicobacter pylori.* Badaniem objęto 50 chorych: 20 z żółciowym nieżytem żołądka, 20 z nadżerkowym zapaleniem żołądka, 10 zdrowych ochotników stanowiących grupę kontrolną. U wszystkich oznaczano w surowicy poziom Il-8, Il-2R, Il-6R. W każdej grupie u połowy chorych stwierdzono infekcję *Helicobacter pylori* testem urazowym. Średni poziom Il-8 u chorych z żółciowym nieżytem żołądka był znacznie wyższy niż w grupie kontrolnej. W grupie dotkniętej infekcją *Helicobacter pylori* poziom był wyższy niż u pacjentów pozbawionych infekcji. W nadżerkowym zapaleniu żołądka z towarzyszącą infekcją *Helicobacter pylori* zanotowano także wyższe poziomy Il-8 niż u chorych bez infekcji. Najwyższe wartości Il-6R obserwowano w żółciowym nieżycie żołądka ze współistniejącym zakażeniem *Helicobacter pylori,* były one znacznie wyższe niż u chorych bez zakażenia. W nadżerkowym zapaleniu żołądka z infekcją *Helicobacter pylori* poziom Il-6R był wyższy niż w grupie bez infekcji (tu pozostawał na poziomie grupy kontrolnej). Nie zaobserwowano wpływu infekcji *Helicobacter pylori* na poziom Il-2R u chorych z żółciowym nieżytem żołądka i nadżerkowym zapaleniu żołądka. Wartości te we wszystkich badanych grupach były podobne jak w grupie kontrolnej. Nasze badania wskazują na to, że w żółciowym nieżycie żołądka stwierdza się wyższe wartości poziomu Il-8 i Il-6R niż w zapaleniu nadżerkowym. Najwyższe poziomy Il-8 i Il-6R obserwowano przy współistnieniu infekcji *Helicobacter pylori.*